REMARKS

Claim 7 is amended herein. Support for the amendment to Claim 7 is found in the specification, for example, at page 17, lines 15-34. No new matter is added by the amendment.

Upon entry of the amendment, Claims 1-12 are presently under examination.

Rejection under 35 U.S.C. §§102/103

Claims 1-6, 11 and 12 are rejected under 35 U.S.C. §102(b) as anticipated by, or, in the alternative, under 35 U.S.C. §103 as obvious over, Keetch *et al.* J. Urol. 152:247-250, 1994.

The Office Action states that Keetch teaches producing nonbacterial prostatitis model mice by injecting ventral prostate homogenate and pertussis toxin into the prostate; that the thus-produced model mice suffer from inflammation, edema, and epithelial cell degeneration; and that the inflammatory process was localized to the lateral and dorsal lobes of the prostate, and that no inflammation was observed in other organs such as the bladder. The Office Action states that, regardless of whether the Keetch model mice were produced by a process different from that of the present invention, the mice may have the same structure because inflammation is locally observed in the dorsal lobes of the prostate.

Applicants submit that the presently claimed nonbacterial prostatitis animal model is distinct from, and, thus, novel over, the mouse model of Keetch. The invention of Claim 1 is characterized in that:

1. Nonhuman animal models are prepared by injecting hydrochloric acid beneath the prostatic capsule;

2. The injected hydrochloric acid has a concentration of 0.1 N to 0.4 N;

3. The resulting animal models exhibit tissue damage in the prostate tissue that is characteristically observed in human chronic nonbacterial prostatitis, and a lower urinary tract disorder that is characteristically observed in human chronic nonbacterial prostatitis; and

4. The resulting animal models are free from disorders in urethra and bladder tissues.

Regarding Item 4, Keetch merely discloses that, "[e]xamination of the other organs harvested - seminal vesicles, testis, coagulation gland, bladder, heart, lungs, and liver - revealed no inflammation." *Keetch* at page 248, right column. In other words, Keetch is silent as to whether or not urethra tissue damage occurred. Thus, Keetch does not teach an animal model having tissue damage in urethral tissue.

Keetch also nowhere discloses the Item 3 feature described above. Keetch states that the development of autoimmune prostate inflammation was observed (see "RESULTS"). In contrast, the data provided in Applicants' specification indicates that the animal models of the invention of Claim 1 not only exhibit severe fibroblast hyperplasia and fibrosis in the interstitium of the prostate; inflammatory cellular infiltration (the disorder characteristically observed in human chronic nonbacterial prostatitis), but also exhibit pollakiuria, reduced effective bladder capacity, and residual urine (obstruction of the lower urinary tract characteristically observed in human chronic nonbacterial prostatitis) (see, for example, Figs. 1, 2, 5 and 6). Keetch is silent as to whether or not the treated mice exhibited pollakiuria, reduced effective bladder capacity, and residual urine. Thus, Keetch does not teach an animal model having the same properties as that of the claimed nonbacterial prostatitis animal model.

As described above, the animal models of Keetch are different from the animal models of the present invention in the method of production thereof, and the observed disorders. Therefore, the animal models of the present invention are neither identical with nor obvious from those of Keetch.

Rejection under 35 U.S.C. §103

Claims 7-10 are rejected under 35 U.S.C. §103 as obvious over Lang, Keetch, Fulmer, Robinette, and Royston in view of Goto. The Office Action states that the claimed methods are obvious because they do not specify that the method results in a nonbacterial animal model with localized inflammation in the prostate without lower urinary tract involvement.

Applicants submit that the claimed methods for preparing a nonbacterial prostatitis animal model are not obvious over the claimed references because no combination of these teachings would lead one to the presently claimed nonbacterial prostatitis model. The presently claimed methods result in a nonbacterial prostatitis animal model that does not have tissue damage in urethral and bladder tissues. As confirmed by the Office Action at page 7, first paragraph, this model is distinct from any teaching in the cited references. Accordingly, the cited references, alone or combined, do not render the invention of Claims 7-10 obvious.

CONCLUSION

In view of the above, Applicants respectfully maintain that claims are patentable and request that they be passed to issue. Applicants invite the Examiner to call the undersigned if any remaining issues might be resolved by telephone.

Please charge any additional fees, including any fees for additional extension of time, or credit overpayment to Deposit Account No. 11-1410.

Respectfully submitted,

KNOBBE, MARTENS, OLSON & BEAR, LLP

Dated: December 16, 2008 By: /Kerry Taylor/
Kerry Taylor
Registration No. 43,947
Attorney of Record
Customer No. 20,995
(619) 235-8550

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